Laryngeal Mask and Pulmonary Edema

To the Editor.—We recently encountered two patients who developed pulmonary edema after insertion of a laryngeal mask. Two otherwise healthy adult patients scheduled for elective orthopedic surgery were anesthetized with a mixture of nitrous oxide, oxygen, and halothane, while breathing spontaneously.

A laryngeal mask (LM) was inserted under deep anesthesia. In both cases difficulties were encountered in inserting the LM and the patients developed signs of airway obstruction with inspiratory stridor and chest retraction. The LM was successfully reinserted in both patients after the third attempt. Despite relieving the airway obstruction, the hemoglobin oxygen saturation was low (90–92% on a FIO2 of 0.5) and wet rales were heard over both lung fields. An arterial blood sample for blood gas analysis was obtained in both patients. In the first case, there was a mild respiratory acidosis and hypoxemia (pH 7.32, PaO2 = 47 mmHg, PaCO2 = 65 mmHg, SaO2 = 90%, FiO2 = 0.4, and spontaneous breathing). In the second case, the blood gas analysis showed severe hypoxemia (pH 7.36, PaO2 = 55 mmHg, PaCO2 = 46 mmHg, SaO2 = 88%, FiO2 = 0.4, while breathing spontaneously). In the postanesthesia care unit, the chest x-ray showed frank pulmonary edema, which was promptly and successfully treated with intravenous furosemide and 100% oxygen by mask. There was no fluid overload in either of the cases. The first patient had received 500 mL of balanced Ringer’s lactate solution during surgery and 100 mL more prior to the chest x-ray, whereas the second patient had received 500 mL during surgery and 120 mL prior to the chest x-ray.

Pulmonary edema has been associated with airway obstruction in children and adults.1,2 The mechanisms causing pulmonary edema induced by airway obstruction are multifactorial and not entirely understood. It is believed that intrathoracic negative pressure created by the inspiratory effort through an obstructed airway will promote an increased venous return to the pulmonary circulation increasing pulmonary capillary hydrostatic pressure. This negative pressure also will decrease pulmonary interstitial hydrostatic pressure, promoting filtration of the fluid from the capillaries into the lung interstitium. Finally, the intense sympathetic discharge triggered by hypoxia will cause increased capillary hydrostatic pressures damaging the alveolar-capillary membrane, increasing permeability and further worsening edema.3

A 10% incidence of malposition of the LM has been reported.4 This could lead to upper respiratory airway obstruction. Forceful spontaneous breathing through an obstructed airway could induce pulmonary edema through the mechanisms described above.

We conclude that, apart from laryngospasm and aspiration of stomach content, malposition of the LM can induce frank pulmonary edema due to upper respiratory airway obstruction.

Tiberiu Ezri, M.D.
Virgil Priscu, M.D.
Peter Szmun, M.D.
David Soroker, M.D.
Department of Anesthesiology
Kaplan Hospital
Rehovot, 76100
Israel

References


(Accepted for publication October 15, 1992)

Preemptive Analgesia or Anoci-Association

To the Editor.—The recent article by Katz et al.1 describing preemptive analgesia is suggestive of an earlier concept proposed by George Crile. Sr. many years ago. Crile introduced the term “anoci-association” as a technique to reduce surgical stress and improve postoperative status. This procedure involved general anesthesia for surgical operations combined with infiltration of local anesthetics to block noxious impulses arising from the surgical wound. In this way, he believed that the patient was protected from the stress of

Anesthesiology. V 78. No 1. Jan 1994
surgery more effectively than by general anesthesia alone. Once more, everything old is new again.

Barry G. Smiler, M.D.
Sarasota Memorial Hospital
1700 South Tamiami Trail
Sarasota, Florida 34239

Reference


(Accepted for publication October 22, 1992.)

Anesthesiology
28:220-221, 1993
© 1993 American Society of Anesthesiologists, Inc.
J. B. Lippincott Company, Philadelphia

In Reply:—We would like to thank Smiler for drawing our attention to the truly remarkable, innovative, and creative work of Dr. George Washington Crile,
(1861-1945), a prolific author of 24 books and more than 100 articles.1 As a surgeon in the late 19th century and early 20th century, when radical surgery was a dominant force in America, Crile pioneered the study of surgical shock, combining laboratory investigations with his surgical practice and his astute clinical observations to improve the safety of surgical procedures.2 His research spanned many fields, and in developing his various theories, he incorporated research findings and concepts from anesthesiology, biology, physiology, psychology, and surgery.

The idea of anoci-association developed out of Crile’s work on shock and exhaustion (fig. 1). Crile believed that both intense fear and noxious stimulation produced shock. Moreover, he assumed that the effects of shock on the central nervous system were identical whether brought about by distressing emotional events or by noxious somatic stimuli. He proposed that shock and exhaustion could be prevented, and the patient's postoperative status improved, by blocking all noxious or harmful (anoci) stimuli (associations) from reaching the brain during the surgical operation. Accordingly, Crile recommended general anesthesia to prevent traumatic emotional experiences from reaching conscious awareness and prevent the_plus_intraoperative local anesthetic infiltrations to prevent noxious surgical inputs from reaching the brain. Together, the administration of these agents provided for what Crile termed the shockless operation through anoci-association.

To achieve complete anoci-association, Crile advocated the use of multiple anesthetic agents and techniques before, during, and after surgery, heralding the current trends in preemptive analgesia and multimodal, balanced analgesia.3,4 In discussing the importance of complete blockade, Crile wrote: “... There is no single agent that alone can produce anoci-association, which is the goal of operative surgery. We, therefore, do not advocate ether alone; nor chloroform alone; nor nitrous-oxide-oxygen alone; we do not advocate local anesthesia alone; nor morphin and scopolamin alone; nor spinal anesthesia alone, but through selection and combination of anesthetics we aim to attain the anesthesia that in the case in hand will exclude all stimuli from the brain, and thereby attain anoci-association” (page 109).1

Crile’s technique for ensuring complete anoci-association included premedication with morphine plus scopolamine, general anesthesia

Fig. 1. Illustration of Crile’s concept of anoci-association. (I) In the conscious patient, auditory, olfactory, and visual input from special sense organs, and noxious somatic impulses from peripheral nociceptors are transmitted to the brain, where they contribute to shock and exhaustion. (II) Under general anesthesia alone, noxious somatic impulses arising from trauma continue to reach the brain. (III) The shockless operation achieved by complete anoci-association. Transmission of noxious auditory, olfactory, and visual impulses are prevented from reaching the brain with use of general anesthesia, and noxious somatic impulses arising from the trauma are blocked by local anesthesia. (Reproduced from Crile and Lower.1)